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te Meerman, Sanne; Batstra, Laura; Hoekstra, Rink; Grietens, Hans

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## Study books on ADHD genetics: balanced or biased?

Sanne te Meerman<sup>a</sup>, Laura Batstra<sup>a</sup>, Rink Hoekstra<sup>b</sup> and Hans Grietens<sup>a</sup>

<sup>a</sup>Department of Special Needs Education and Child Care, University of Groningen, the Netherlands; <sup>b</sup>Department of Educational Sciences, Learning & Instruction, University of Groningen, the Netherlands

### ABSTRACT

Academic study books are essential assets for disseminating knowledge about ADHD to future healthcare professionals. This study examined if they are balanced with regard to genetics. We selected and analyzed study books (N=43) used in (pre) master's programmes at 10 universities in the Netherlands. Because the mere behaviourally informed quantitative genetics give a much higher effect size of the genetic involvement in ADHD, it is important that study books contrast these findings with molecular genetics' outcomes. The latter studies use real genetic data, and their low effect sizes expose the potential weaknesses of quantitative genetics, like underestimating the involvement of the environment. Only a quarter of books mention both effect sizes and contrast these findings, while another quarter does not discuss any effect size. Most importantly, however, roughly half of the books in our sample mention only the effect sizes from quantitative genetic studies without addressing the low explained variance of molecular genetic studies. This may confuse readers by suggesting that the weakly associated genes support the quite spectacular, but potentially flawed estimates of twin, family and adoption studies, while they actually contradict them.

### KEYWORDS

ADHD; medicalization; genetics; twin studies; genes

## Introduction

Although there is substantial variability between countries, worldwide an estimated 5–7% of school-aged children are diagnosed with attention deficit hyperactivity disorder (ADHD), which makes ADHD the most often used psychiatric classification assigned to children (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007). ADHD and other mental disorders are defined in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM). According to the authors of the fourth edition (American Psychiatric Association, 1994), these disorders are “valuable heuristic constructs” useful for research and practice but they are not “well-defined entities that describe nature exactly as it is” (Frances, First, & Pincus, 1995, p. 12). The fifth edition of the DSM however, lists the disorder as “neurodevelopmental” (American Psychiatric Association, 2013). And, in an influential consensus statement released in 2002, causing much disagreement and dismay (Timimi, 2004; Whitely, 2015), several opinion leaders claim there is no “substantial scientific disagreement over whether ADHD is a real medical condition” (Barkley et al., 2002, p. 96).

One of the pillars of this medical view is the concept of heritability that is calculated using the “observed and expected resemblance” of twins and

relatives (Visscher, Hill, & Wray, 2008). According to the DSM-5, “the heritability of ADHD is substantial” (American Psychiatric Association, 2013) and the consensus statement states it is “nearly approaching the genetic contribution to human height” (Barkley et al., 2002, p. 97). Measuring complex human behaviour is far more difficult than assessing height, yet several researchers claim these twin studies indicate that “60–90% of current phenotypic variance can be explained by inherited factors” (Stergiakouli, 2010, p. 552). However, molecular studies into genetics show only small effects for individual genes associated with ADHD. Aggregated they account for less than 10% of explained variance (Franke, Neale, & Faraone, 2009). Interestingly, the consensus statement mentions an associated gene but the effect size of the association is unspecified.

The practice of mentioning only associations has often been criticized. For instance, the commonly used p-values are based on *average* differences between groups, thus largely ignore that there can still be substantial overlap between groups. Measures of effect size (ES, plural: ESs) provide insight into the amount of overlap and display information about the magnitude of a result, unlike p-values (Ferguson, 2009; Thompson, 1999).

We consider the explained variance according to twin, family and adoption studies as an example of ES, since they provide an indication of the size (and as a

result, of the importance) of genetics' contribution to ADHD-related behaviour. However, we argue that mentioning the ESs of molecular genetics—in addition to mentioning the ES of twin, family and adoption studies—is paramount for several reasons. First, twin, family and adoption studies provide less “hard” evidence for the genetic origins of behaviour. These “quantitative” or “quantitative genetic” studies, as they are often called, use behavioural information only to estimate genetic influence. Molecular genetic studies relate behavioural information to genetic material of test subjects. Hence, including the ESs of molecular studies seems important for any writer aiming to shed light on the genetics of ADHD related behaviours. Second, quantitative genetic studies are prone to rating-bias because “parents rating twins’ activity levels tend to emphasize differences between fraternal twins but similarities between identical twins, leading to an inflated heritability estimate” (Nigg, 2006, p. 198). Third, there are several assumptions underlying the heritability estimate that are under debate. For instance, “the assumption that genetic and environmental influences are independent” (Johnson, Penke, & Spinath, 2011, p. 258) does not hold when genes and environments correlate and interact with each other. Furthermore, the “equal environment assumption”, positing that fraternal and identical twin pairs have similar environments, is often scrutinized, for instance because “twins develop in different types of placental environments” (Freitag, Rohde, Lempp, & Romanos, 2010, p. 314).

According to Johnson et al. (2011, p. 255) “the limitations of heritability estimates for understanding underlying biology have long been known to behaviour geneticists but not necessarily to the many social scientists who are becoming newly interested in the presence of genetic influences on behavioural traits”. For students just learning about the intricacies of genetics, mentioning the low (aggregated) ESs found by molecular genetic studies can help to reveal potential weaknesses of quantitative genetic studies while simultaneously providing insights into the relations of genetics and environment. On the other hand, drawing attention to quantitative genetics studies and their quite spectacular high estimates of heritability while disregarding the low (aggregated) ESs of molecular genetic studies can obfuscate such weaknesses and might easily suggest a stronger ES of certain genes than the ESs actually observed. High heritability estimates in combination with unspecified associations—followed by an impressive array of genes with elusive names such as *TPH-2*, *SLC6A4*, *CHRNA4* and *GRIN2A* (Faraone & Mick, 2010) might lead students to believe that these weakly associated genes explain these high

heritability findings and the problematic behaviour, while their explanatory power is very limited.

In this study, we analyse if and how authors of study books used at Dutch universities mention the ESs of both quantitative and molecular genetic studies and how they name and explain difference between them to students, or fail to do so. A thoughtful consideration of the ways study books differ in this respect might help authors and teachers alike to avoid confusion and provide meaningful insights to those “newly interested” in ADHD.

## Methodology

### Data selection

Out of all 18 Dutch public universities, those that are known to have a wide orientation, including behavioural and often medical programmes, were selected ( $n = 10$ ). One university focuses on distance education. The non-selected universities are (mostly) technical in orientation (three), theological (four) or agricultural (one). The Netherlands also has three privately funded universities that were not included (Source: Association of Universities in the Netherlands, [www.vsnul.nl](http://www.vsnul.nl)). From the selected universities, the minor's, bachelor's, pre-master's and master's programmes were selected for further study. Forty-three study books were selected from the following fields of study: psychopathology, (biological, cognitive, clinical, biological) psychology, psychiatry, psychiatric disorders, diagnostics and behavioural problems. Of these study books, we selected the most up-to-date versions published in or before January 2016. This “purposeful sampling” (Coyne, 1997, p. 623) aimed to select contemporary state-of-the-art academic study books aimed at educating professionals that are likely to hold mental healthcare related key-positions in their future. Additionally, because many of the English books used can be found in university libraries worldwide, they are likely to be used in academic courses at other universities.

The glossaries of mandatory study books specified in the study guides were searched for the keywords “ADHD” and “attention deficit hyperactivity disorder”. Books with either a dedicated section/chapter on ADHD or a dedicated chapter on psychopathology that included ADHD in a subsection or paragraph were selected.

### Analytic framework

Table I displays the framework deployed to categorize the data in our study set. It consists of four categories, each representing a combination of authors' mentioning or omitting ESs of either quantitative or molecular genetic studies.

**Table I.** Criteria for classifying ES in study books.

|                                     |  | Molecular genetic studies  |   |
|-------------------------------------|--|--|---|
|                                     |  | Omitted: No involved genes mentioned, or only involved genes mentioned, but no ES specified, verbally or numerically   | Mentioned: ES mentioned verbally and/or numerically   |
| <b>Quantitative genetic studies</b> | <b>Omitted: No ES mentioned verbally or numerically</b>  | (A) Superficial: no reference to explained variance of individual or aggregated genes, and no estimate of heritability of family/twin or adoption studies.                       | (B) Molecular genetics only: reference to explained variance of individual or aggregated genes, but not to heritability according to family/twin or adoption studies. |
|                                     | <b>Mentioned: ES as range (60–90), exact number (0.76), or verbally qualified (high/low, etc.)</b> | (C) Quantitative studies only: reference to heritability according to family/twin or adoption studies, but no reference to explained variance of individual or aggregated genes. | (D) Elaborate: reference to explained variance of individual or aggregated genes, and reference to heritability according to family/twin or adoption studies.         |

**Category A: the “superficial” study books**

Study books fell into this category if they contained no ESs from both quantitative and molecular genetic studies.

**Category B: the “molecular genetics only” study books**

This category was populated by study books that mention ESs of molecular genetic studies, but do not mention ESs of quantitative studies.

**Category C: the “quantitative genetics only” study books**

Study books that mentioned only the ESs of quantitative, but not ESs from molecular genetic studies were placed in this category.

**Category D: the “elaborate” study books**

This category was populated by study books that were considered to be “elaborate” as they contained ESs from both quantitative and molecular genetic studies.

**Criteria**

In quantitative studies, any odds-ratio based on ADHD prevalence of identical versus fraternal twins, heritability estimate, range of heritability, or verbal qualification such as high or low was considered as a mention of ES. Any relative/ordinal association ( $A > B$ ) was not considered as such. For instance, stating that identical twins concord more often than non-identical twins was not scored as mentioning an ES.

Relating to molecular genetic studies, any verbally/numerically specified association of any or several genes was considered as a mention of ES, while any unspecified association was considered as an omission of ES. For instance, statements that genes cause attention problems in many/few cases were considered as verbal suggestion of an ES. On the other hand, the pronoun “some” (for instance “in some cases ADHD might be caused by”) was not considered as specified. Claims about genes that *may be involved* were also considered as unspecified, so omitted ESs. Table I displays the criteria.

In addition to these criteria, other factors were considered that give a deeper insight into the different strategies used by authors to explain genetics, such as the size of a section, use of contrast when explaining the difference between quantitative and molecular genetics and the precision of ESs given. These additional parameters might provide insights into the choices that authors make to explain the genetics of ADHD-related behaviours.

We have selected quotes from at least two books in each section to illustrate both a typical book in the category and—if possible—a less typical one. This approach provides openness about the categorization, the boundary cases and the cut-off points. We also included older versions of books if this provided additional insights. The comparison between editions enabled us to identify changes and reflect on choices made by authors.

**Results**

Table II gives an overview of the frequencies with which authors chose the four different ways of displaying ESs. For reasons of replicability we included an overview in Appendix A, classifying and referencing the study books in our sample.

**A: superficial**

About 23% of the authors are *superficial* relating to ESs of genetic studies. They do not mention ESs or genetic influence in general. These are often the smaller sections in books, on average about five pages. For instance, Bernstein, Penner, Clarke-Stewart, and Roy (2012, p. 631) write:

ADHD may result from a genetic predisposition. Some studies suggest that the genes involved may be those that regulate dopamine, a neurotransmitter important in the functioning of the attention system (...).<sup>1</sup> Other factors, including (...). Exactly how all these factors combine is still not clear.

This is considered a “typical” example of books in this category: several etiological factors are



**Table II.** Specificity of ES (quantitative/molecular genetics) in study books.

| Twin studies |  | Molecular genetics studies  |  |
|--------------|--|---|--|
|              |  | Omitted: No involved genes mentioned, or only involved genes mentioned, but no ES specified, verbally or numerically                        | Mentioned: ES mentioned verbally and/or numerically  |
|              | <b>Omitted: No ES mentioned verbally or numerically</b>  | A: superficial<br>$n = 10$ (23%) <sup>a</sup><br>Average: 5 pages <sup>b</sup><br>Specific genes: $n = 1$ <sup>c</sup><br>(10% of category) | B: molecular genetics only<br>$n = 3$ (7%)<br>Average: 15 pages<br>Specific genes: $n = 2$<br>(100% of category)                       |
|              | <b>Mentioned: ES as range (60–90), exact number (0.76), or verbally qualified (high/low, etc.)</b> | C: quantitative studies only<br>$n = 20$ (47%)<br>Average: 7 pages<br>Specific genes: $n = 8$<br>(38% of category)                          | D: elaborate<br>$n = 10$ (23%)<br>Average: 16 pages<br>Specific genes $n = 10$<br>(100% of category)<br>Contrast: $n = 9$ <sup>d</sup> |

<sup>a</sup>Percentage of total.<sup>b</sup>Average number of pages of books in category.<sup>c</sup>Specific gene(s) mentioned by authors (by name, e.g., DRD4) or by function (e.g., dopamine related).<sup>d</sup>Contrast between ES of quantitative vs. molecular genetic studies explicated.

mentioned, including genetics, but the effects are not specified. Likewise, the next example also indicates several causal factors and refers to their possible interaction, without claims of ES:

Twin studies show a greater incidence of ADHD among identical twins than non-identical twins. Studies have compared the incidence of ADHD among children whose parents are biologically related to the child with the incidence of the children of parents where the child was adopted. These indicate a greater probability of ADHD appearing in biologically related parents and children. (Farrell, 2012, p. 152)

With 11 pages, this chapter is relatively large considering the size of the other ADHD sections in this category. The author mentions twin and adoption studies, but does not discuss their ESs nor those of genes.

### **B: molecular genetics only**

Two books in our sample are classified as “molecular genetics only”, referring only to molecular genetic studies ESs. One of them is Stahl’s essential psychopharmacology (Stahl, 2013). With 32 pages on ADHD, this is one of the largest chapters on ADHD. With regard to genetics, there is no reference to quantitative studies, which is a departure from the earlier edition (Stahl, 2008), that does mention a heritability of ~75%. In the 2013 version, the authors do refer to genes:

genes that code for subtle molecular abnormalities are thought to be just as important to the etiology of ADHD as they are to the etiology of schizophrenia. (p. 480)

And later in the text:

The major genes implicated in ADHD are those linked to the neurotransmitter dopamine. (p. 480)

We consider “just as important” as a reference to an ES for the molecular studies, because it is named as “important”. The text contains no further information about the magnitude of the effect. Because the author refers to the chapter on schizophrenia, we have searched this chapter for ESs. Here, the author is slightly more elaborate on twin studies and heritability but does not give a heritability estimate of schizophrenia (or ADHD).

The best evidence that the environment is involved in schizophrenia is that only half of identical twins of patients with schizophrenia also have schizophrenia. (Stahl, 2013, p. 115)

Interestingly, the concordance between twins is used by the authors to express caution about the importance of heritability. Regarding molecular genetics, the ES of genes on the molecular level is not mentioned, yet the authors downplay a deterministic effect of abnormal genes and suggest a pathway for normal genes to result in abnormal behaviour by mediation of the environment.

Thus, mental illnesses are due not only to genes that are abnormal in their DNA and in the function of the proteins they code, but also to normal genes that make normal functioning proteins but are activated or silenced at the wrong times by the environment. (Stahl, 2013, p. 114)

This is meaningful information about molecular genetics and effects sizes. However, we argue that the authors made a rather unfortunate choice by suggesting an “important” effect of “genes that code for subtle molecular abnormalities”, without explaining this any further in the chapter about ADHD or the chapter about schizophrenia that they are referring to.

Another book we classified to be in this category is by Barlow and Durand (2015). The authors mention no explicit ES in a paragraph where family studies are discussed, but claim ADHD is “highly influenced by

genetics” in the next paragraph, referring to molecular genetics’ findings.

Research on ADHD (and on other disorders) is finding that in many cases, however, mutations occur that either create extra copies of a gene on one chromosome or result in the deletion of genes (called copy number variants—CNVs) (...) the additions or deletions of one or more genes result in disrupted development. (Barlow & Durand, 2015, p. 517)

Although some copy number variants (CNVs) occur relatively often in groups of those diagnosed with ADHD, most are rare in the general population as well as in the population of those diagnosed. For instance, a duplication on the 15th chromosome at location 15q13.3 has a frequency of 1.25% in the research group of those diagnosed with ADHD. Although this is about two times higher than in the general population (0.6%) (Williams et al., 2012), it does not contribute much to the heritability of ADHD in general and many with the duplication do not display ADHD behaviours. The point that Barlow & Durand do not make relating to CNVs is that “the ESs of this and previous studies are quite small” (Martin, O’Donovan, Thapar, Langley, & Williams, 2015, p. 3). The “disrupted development” related to CNVs is therefore only probabilistic and not as deterministic as suggested.

### C: quantitative genetics only

About half of our set of books mentioned ESs from quantitative research without mentioning ESs of molecular genetic studies. The sections were on average slightly larger than those in the “superficial” category (average: seven pages).

An example in this category that stands out is from a Dutch study book (Hengeveld & Van Balkom, 2009).

Around 80% of variation in hyperactivity, impulsivity and concentration weakness between children is based on heritable factors. The risk for ADHD for brothers/sisters of a child with ADHD is 3–5 times higher compared to the normal population. In second-degree relatives the risk is 2 times higher. This indicates multifactorial heritability where a number of risk-genes in combination drive the heritable influence. The involvement of a number of candidate genes from the dopaminergic neurotransmission (dopamine-D4- and -D5-receptor and dopamine transporter) in ADHD is confirmed in meta-analysis. (Buitelaar & Van der Gaag, 2009, p. 544)<sup>2</sup>

This book mentions a rather precise heritability estimate of “around 80%”. This is not uncommon in this category ( $n = 8$ ) although several books portray heritability as a range ( $n = 9$ ), which is arguably preferable as it avoids creating the misperception that there is one true value for a disorder or trait as the explained variance in fact depends on place and time

(Rutter & Plomin, 1997). More importantly, this book is the only one in our sample that does not relate a numerical heritability estimate to the type of study on which the estimate is based. This is relevant as the risk of confusing ESs of molecular genetic studies and quantitative studies is real. The authors then mention the “involvement of a number of candidate genes”, and explicitly mention the genes’ names but not their ESs, nor the small aggregated ESs of all associated genes. Eight other books in this category explicitly mention genes but not their ESs.

Another potentially confusing example comes from Carter (2014, p. 246).

ADHD tends to run in families and in most cases genetic inheritance, probably involving many genes, is thought to be the most probable underlying cause.

In “most cases genetic inheritance (...) is thought to be the cause” seems like a reference to an ES. While an informed reader might infer that twin/family studies must also be at the basis of the claim about “genetic inheritance”, the empirical basis is in fact unclear.

### D: elaborate

Sections that were most elaborate on both quantitative and molecular genetic studies, were on average relatively large (average: 15 pages). Ninety per cent of books in this category ( $n = 9$ ) contrasted or actively emphasized the difference between population and molecular findings.

An exemplary series of books in this category is Rutter’s *Child and Adolescent Psychiatry*. We cite both from the fifth (Rutter et al., 2008) and the sixth (Thapar et al., 2015) editions. The 2008 version is the most elaborate on genetics within the chapter about ADHD and hyperkinetic disorder:

According to twin studies, ADHD is amongst the most heritable conditions with estimates between 60 and 90%. (...) In contrast to the high heritability estimates, the effects of specific genes are small. When aggregated, they account for only a fraction of variance in symptom expression. How can this gap be explained? First .. (...), Second (...). (Taylor & Sonuga-Barke, 2008, p. 526)

Referring to twin studies, the authors display a range of heritabilities, contrast these findings with the much lower explained variation according to molecular studies, and name four possible explanations for this difference:

- (1) Twin studies overestimating genetic main effects and subsuming the effects of gene X environment interactions.
- (2) The possibility of genes still to be found.
- (3) Interaction effect between genes.

- (4) The potential heterogeneity of the disorder, making several pathways of “genes (and environments)” possible to create “ADHD in different groups of ADHD children” (p. 526).

Especially the way authors discuss the influence of interplay effects on the estimation of heritability makes this chapter stand out. Only one of the other books does this so explicitly; Wicks-Nelson (2015) actually refers to this chapter as she points to gene-environment interaction:

It is important to note that heritability estimates include the effects of gene-environment interaction. (Taylor & Sonuga-Barke, 2008)

The difference between this edition (Rutter et al., 2008) and the new edition that appeared in 2015<sup>3</sup> is interesting (Thapar et al., 2015).

It is pretty clear that genetic influences are involved. The strongest evidence comes from studies comparing the similarity of monozygotic and dizygotic twins: genetic influences account for 70–90% of the variance in different studies, on the (reasonable) assumption that environmental factors influence both twins more or less equally. Data from research has not, however, established an unequivocally causative role for individual genetic variants (...). Several molecular genetic variants are known to be associated, but their effects are small and the causal pathways are not established. (Thapar et al., 2015, p. 742)

Note that the authors mention a smaller heritability range compared to the earlier edition (70–90% instead of 60–90%). In this edition they also briefly discuss the “equal environment assumption”—the only study book in our sample that does so. This assumption is fiercely debated by Joseph (2006) and Furman (2008). If identical twins select more similar environments than fraternal twins as these critics argue, this might be another cause for an inflated heritability estimate.

Another noteworthy observation is that this is the only book in our sample that explicitly refers to another chapter, when discussing interplay effects.

Genetic and environmental influences are profoundly intertwined for ADHD and need to be considered jointly (see Chapter 24). (Thapar et al., 2015, p. 742)

Perhaps this choice was made because the principles of genetics apply to several other disorders as well. Several books include a chapter about genetics, but do not refer to it when discussing ADHD. This book is among the most elaborate on this topic, with a separate chapter on genetics and another on epigenetics.

## Discussion

Discourse studies like ours can be critiqued because they do not “necessarily indicate that it is being read and comprehended in the same way from one reader to the next” (Freedman, 2015, p. 39), and different researchers likewise might have different interpretations. Additionally, the analysed discourse does not stand alone as other domains of discourse (Ferri, Connor, Solis, Valle, & Volpitta, 2005), and other unselected sections and chapters can shape the perception of readers as well.

We have aimed to counter these flaws by using a straightforward and descriptive approach that is less sensitive to interpretation. This makes analysing a relatively large selection of study books feasible, which facilitates comparison and inference. After Switzerland and Sweden, the Netherlands has most universities in the top 100, when related to population size (source: <http://www.economist.com/news/special-report/21646987-competition-among-universities-has-become-intense-and-international-top-class>). This indicates that the academic proficiency of our selection of study books used in Dutch universities is likely a good representation of the “state of the art” of study books that discuss ADHD.

Returning to the research question, our research indicates that not many authors of study books explicitly mention and relate the outcomes of quantitative as well as molecular genetics. In this section, we will reflect on these findings and discuss how teachers as well as authors of study books and research articles might avoid confusion about the genetics of ADHD.

About a quarter of the study books in our sample were unspecific about quantitative and molecular genetic studies’ effect size. Possibly, this was a strategy of *hedging*. Viewed positively, hedging is a means to project “honesty, modesty and proper caution” (Swales, 1990, p. 175). Other scholars are less positive and regard hedges as phrases or words “whose job it is to make things more or less fuzzy” (Lakoff, 1973, p. 471). Regardless of a positive or critical view on hedging, when there is not much space for an elaborate discussion authors understandably need to simplify. In such cases, caution—even at the risk of becoming somewhat fuzzy—is arguably a better option than over-simplifying or becoming biased.

Few authors ( $n = 2$ ) exclusively mention ESs of molecular studies. These studies are far less positive on the genetic contribution to ADHD and might actually reveal some of the weaknesses of quantitative studies. Unfortunately, the authors suggest a considerable contribution from individual genes. Stahl (2013) does seem to avoid the suggestion that the effect of genes is absolute, by explicitly stating that



“normal” genes can cause abnormal behaviour by environmental activation—which is a very implicit referral to effect size. However, Stahl only does so outside the chapter on ADHD, after suggesting that ADHD genes are thought to be “just as important for ADHD as for schizophrenia”.

More troubling is the phrasing by Barlow and Durand (2015, p. 517) relating to copy number variation (CNV). They state that in “many cases (...) the additions or deletion of one or more genes result in disrupted development”. The authors perhaps followed their empirical source. Elia et al. (2010, p. 637) claim ADHD is a “highly heritable disorder” and write that they “identified 222 inherited copy number variations (CNVs) within 335 ADHD patients and their parents that were not detected in 2026 unrelated healthy individuals” (Elia et al., 2010, p. 637). This ad hoc discovery of 222 inherited genetic variations in the “patients” is not likely to be the cause of their behaviour. A similar set of CNVs, but unique for the control group, could be identified in the control group and is just as likely to be (un)related to their behaviour. The researchers merely used the set of genes to statistically test for an association with candidate genes. They indeed found a significant association with weak effect size that does not justify their claim that their results “suggest that rare inherited structural variations play an important role in ADHD development” (Elia et al., 2010, p. 637). This way, the researchers themselves suggested the effect size was strong, which might have confused the authors of the study book.

The most striking finding in our dataset, however, was the high occurrence of books that mention the high effect sizes of quantitative studies while remaining vague about the effect size of molecular studies. For instance, Bear, Connors, and Paradiso (2016, p. 724) states: “genes related to the function of dopaminergic neurons have been reported to be abnormal in people with ADHD”. This is arguably a form of publication bias and gives readers the wrong impression that these genes actually explain the high heritability.

As with Barlow and Durand, these selective writings might have been influenced themselves by the discourse surrounding ADHD. According to Hyland (1998, p. 358) “in academic writing, the choices individuals make are socially shaped and constrained by the possibilities made available to them by the discourse conventions of their disciplines”. Perhaps this is a self-reinforcing phenomenon in which empirical research and documents such as the consensus statement discussed earlier (Barkley et al., 2002) are also involved. The statement also failed to mention the effect size of the “one gene (...) associated with this disorder” (Barkley et al., 2002, p. 97) and this might tacitly shape the possibilities within the discourse community and justify that this is an appropriate

way to write about genetics in study books. Although the contrast between the effect sizes of quantitative and molecular genetic studies was not named yet as “the missing heritability problem” in 2002 (this term was introduced by Maher, 2008), and much molecular research into ADHD was still to be done, the selective writing of the consensus statement might have shaped a precedent. In fact, the statement is reprinted (but not the reply to it) in a study book that mentions the ES of quantitative studies in combination with unspecified associations of several genes mentioned explicitly (Kerig, Ludlow, & Wenar, 2012). Furthermore, the book seems to generalize the associated genes even stronger than Bear et al. (2016) by stating “children with predominantly inattentive ADHD have changes to their norepinephrine transporter gene” (p. 223).

The notion that authors not only reach many students, but also inspire each other in the way they tackle a subject such as ADHD, shows the importance of scrutinizing other domains of discourse relating to ADHD—such as empirical studies and psycho-education on the internet. Furthermore, selective writings can obscure important information for fellow and future professionals about the nosological and research related challenges of social science and psychiatry and possible solutions such as suggested by, for instance, Van Der Sluis, Verhage, Posthuma, and Dolan (2010). Perhaps worse, the suggestion of a rather clear-cut genetic affliction inside the child might obscure opportunities to help schools and families adapt to the child’s needs and might also cause lack of consideration for cultural and other points of view.

To end on a positive note: although much can be said about the industry-sponsored follow-up on the consensus statement (Kooij et al., 2010), it does mention both effect sizes from quantitative as well as molecular studies, albeit without highlighting the contrasting findings. And in our data-sample, roughly a quarter of the ADHD sections were classified as “elaborate”, explicating and often contrasting effect sizes of both quantitative and molecular studies. Heritability is a “slippery” (Jaffee & Price, 2011, p. 276) and “admittedly difficult” concept, even to fierce critics like Joseph (2004, p. 137). As Taylor and Sonuga-Barke (2008) illustrate, contrast can serve as a dialectical tool to give perspective and address larger issues such as the heterogeneity in the group of those diagnosed with ADHD, the limitations of twin studies and gene–environment interplay. Particularly the latter seems a promising perspective. Considering, for instance, that one of the most “important” genetic variations related to ADHD, the DRD4 7-repeat allele, might have been around for 50,000 years and has apparently been quite successful (Wang et al., 2004), it might be time to release the burden from our

children's genes and instead target our culture for research and therapy.

## Notes

1. To avoid confusion, references have been omitted from quotes.
2. All translations: S.t.M.
3. The 2008 edition was not used in our quantitative overview of the findings as we have only used the most recent editions of books for this purpose.

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## Appendix A: Data sample and classification

**Table A1.** Study books and their mentioning of ES.

|  |   |
|--|---|
| <b>Superficial books<br/>(n = 10)</b>      | <p>Anderson, V., Northam, E., Hendy, J., &amp; Wrennall, J. (2001). <i>Developmental neuropsychology: A clinical approach</i> (1st ed.). Philadelphia, PA: Taylor &amp; Francis.</p> <p>Belsky, J. (2013). <i>Experiencing the lifespan</i> (3rd ed.). New York, NY: Worth Publishers.</p> <p>Bernstein, D., Penner, L.A., Clarke-Stewart, A., &amp; Roy, E. (2012). <i>Psychology</i> (9th international ed.). Boston, MA: Cengage Learning.</p> <p>Comer, R. J. (2013). <i>Fundamentals of abnormal psychology</i> (7th ed.). New York, NY: Worth Publishers.</p> <p>Farrell, M. (2012). <i>Educating special children: An introduction to provision for pupils with disabilities and disorders</i> (2nd ed.). New York, NY: Routledge.</p> <p>Gray, P., &amp; Bjorklund, D. F. (2014). <i>Psychology</i> (7th ed.). New York, NY: Worth Publishers.</p> <p>Kolb, B., &amp; Whishaw, I. Q. (2015). <i>Fundamentals of human neuropsychology</i> (7th ed.). New York, NY: Worth Publishers.</p> <p>Ruijsenaars, A. J. J. M., &amp; Ghesquière, P. (1999). <i>Neuropsychologische aspecten van problemen op school</i> (1st ed.). Belgium: Acco, Leuven.</p> <p>Tak, J. A., Bosch, J. D., Begeer, S., &amp; Albrecht, G. (2014). <i>Handboek psychodiagnostiek voor de hulpverlening aan kinderen</i> (8th ed.). Utrecht: De Tijdstroom.</p> <p>Van der Wolf, K., &amp; Van Beukering, T. (2009). <i>Gedragsproblemen in scholen: Het denken en handelen van leraren</i> (1st ed.). Leuven: Acco.</p>   |
| <b>Molecular genetics only<br/>(n = 3)</b> | <p>Barlow, D., &amp; Durand, V. (2015). <i>Abnormal psychology: An integrative approach</i> (7th ed.). Stamford, CT: Cengage Learning.</p> <p>Durand, V., &amp; Barlow, D. (2013). <i>Essentials of abnormal psychology</i> (6th ed.). Stamford, CT: Cengage Learning.</p> <p>Stahl, S. M. (2013). <i>Stahl's essential psychopharmacology: Neuroscientific basis and practical applications</i> (4th ed.). New York, NY: Cambridge University Press.</p>   |
| <b>TF studies only (n = 20)</b>            | <p>Bear, M. F., Connors, B. W., &amp; Paradiso, M. A. (2016). <i>Neuroscience: Exploring the brain</i> (4th ed.). Baltimore, MD: Lippincott Williams &amp; Wilkins.</p> <p>Berk, L. E. (2013). <i>Child development</i> (9th international ed.). New Jersey, NJ: Pearson Education.</p> <p>Braet, C., &amp; Bögels, S. M. (2014). <i>Protocolaire behandelingen voor kinderen en adolescenten met psychische klachten</i> (2nd ed.). Amsterdam: Uitgeverij Boom.</p> <p>Bukatko, D., &amp; Daehler, M. W. (2012). <i>Child development: A thematic approach</i> (6th international ed.). Belmont, CA: Wadsworth.</p> <p>Carter, R. (2014). <i>The brain book</i> (2nd ed.). London: Dorling Kindersley.</p> <p>Delfos, M. F. (2009). <i>Ontwikkelingspsychopathologie</i> (7th ed.). Amsterdam: Pearson.</p> <p>Gazzaniga, M., Heatherton, T., &amp; Halpern, D. (2016). <i>Psychological Science</i> (5th international student ed.). New York, NY: W. W. Norton, Inc.</p> <p>Gleitman, H., &amp; Gross, J. (2011). <i>Psychology</i> (8th international student ed.). New York, NY: W. W. Norton &amp; Company.</p> <p>Hengeveld, M. W., &amp; Van Balkom, A. J. L. M. (2009). <i>Leerboek psychiatrie</i> (2nd ed.). Utrecht: De Tijdstroom.</p> <p>Nevid, J. S., Rathus, S. A., &amp; Greene, B. (2016). <i>Psychiatrie: Een inleiding</i> (8th ed.). Amsterdam: Pearson.</p> <p>Swaab, H., Bouma, A., &amp; Hendriksen, J. (eds) (2011). <i>Klinische kinderneuropsychologie</i> (1st ed.). Amsterdam: Boom.</p> <p>Kerig, P., Ludlow, A., &amp; Wenar, C. (2012). <i>Developmental psychopathology</i> (6th ed.). New York, NY: McGraw-Hill Education.</p> <p>Kolb, B., &amp; Whishaw, I. Q. (2014). <i>An introduction to brain and behavior</i> (4th ed.). New York, NY: Worth Publishers.</p> <p>Nolen-Hoeksema, S. (2014). <i>Abnormal psychology</i> (6th international student ed.). New York, NY: McGraw-Hill Education.</p> <p>Siegler, R. S., DeLoache, J. S., Eisenberg, N., &amp; Saffron, J. (2014). <i>How children develop</i>. New York, NY: Worth Publishers.</p> <p>Smeets, G., Bos, E. R., van der Molen, H. T., &amp; Murre, P. (2009). <i>Klinische psychologie: Diagnostiek en therapie</i> (2nd ed.). Groningen, the Netherlands: Wolters-Noordhoff.</p> <p>Van Lieshout, T. (2009). <i>Pedagogische adviezen voor speciale kinderen</i> (2nd ed.). Houten, the Netherlands: Bohn Stafleu van Loghum.</p> <p>Vandereycken, W., Hoogduin, C. A. L., &amp; Emmelkamp, P. M. G. (2008). <i>Handboek psychopathologie: Deel 1 basisbegrippen</i>. Houten, the Netherlands: Bohn Stafleu van Loghum.</p> <p>Verschueren, K., &amp; Koomen, H. (2008). <i>Handboek diagnostiek in de leerlingenbegeleiding</i> (1st ed.). Antwerpen: Garant.</p> <p>Yeates, K. O., Ris, M. D., Taylor, H. G., &amp; Pennington, B. F. (2010). <i>Pediatric neuropsychology: research, theory, and practice</i>. (2nd ed.). New York, NY: Guilford Publications.</p> |
| <b>Elaborate (n = 10)</b>                  | <p>Carlson, N. R. (2013). <i>Physiology of behavior</i> (11th international ed.). New Jersey, NJ: Pearson Education.</p> <p>Carr, A. (2016). <i>The handbook of child and adolescent clinical psychology: A contextual approach</i> (3rd ed.). New York, NY: Taylor &amp; Francis.</p> <p>Doreleijers, T. A. H., Boer, F., Huisman, J., &amp; Haan, E. H. F. (eds) (2013). <i>Leerboek psychiatrie kinderen en adolescenten</i> (2nd ed.). Utrecht: De Tijdstroom.</p> <p>Farah, M. J., &amp; Feinberg, T. E. (2006). <i>Patient-based approaches to cognitive neuroscience</i> (2nd ed.). Cambridge, MA: MIT Press.</p> <p>Higgins, E. S., &amp; George, M. S. (2013). <i>Neuroscience of clinical psychiatry: The pathophysiology of behavior and mental illness</i> (2nd ed.). Philadelphia, PA: Wolters Kluwer Health.</p> <p>Prins, P. J. M., &amp; Braet, C. (2014). <i>Handboek klinische ontwikkelingspsychologie</i> (2nd ed.). Houten: Bohn Stafleu van Loghum.</p> <p>Thapar, A., Pine, D. S., Leckman, J. F., Scott, S., Snowling, M. J., &amp; Taylor, E. A. (2015). <i>Rutter's child and adolescent psychiatry</i> (6th ed.). Chichester: John Wiley &amp; Sons.</p> <p>Verhulst, F. C. (2015). <i>Leerboek kinder—En jeugdpsychiatrie</i> (4th ed.). Assen: Koninklijke Van Gorcum.</p> <p>Verhulst, F. C., Verheij, F., &amp; Danckaerts, M. (2014). <i>Kinder—En jeugdpsychiatrie</i> (1st ed.). Assen: Koninklijke Van Gorcum.</p> <p>Wicks-Nelson, R. (2015). <i>Abnormal child and adolescent psychology with DSM-5 update</i> (8th ed.). New York, NY: Taylor &amp; Francis.</p>  |